COMPARISON OF ULTRASOUND-GUIDED FOAM SCLEROTHERAPY COMBINED WITH SAPHENO-FEMORAL LIGATION AND SURGICAL TREATMENT OF VARICOSE VEINS

A STUDY OF 50 CASES

DISSERTATION SUBMITTED FOR THE DEGREE OF

MASTER OF SURGERY

BRANCH – I (GENERAL SURGERY)

MARCH - 2009

THE TAMILNADU
DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

CERTIFICATE

This is to certify that the dissertation entitled, **"COMPARISON OF ULTRASOUND GUIDED FOAM SCLEROTHERAPY COMBINED WITH SAPHENOFEMORAL LIGATION AND SURGICAL TREATMENT OF VARICOSE VEINS"** submitted by **Dr.PRATHIBA.N** to the Tamil Nadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.S. Degree Branch-I (General Surgery) is a bonafide research work were carried out by her under direct supervision & guidance.

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DECLARATION

I, **Dr. PRATHIBA. N** solemnly declare that this dissertation titled **"COMPARISON OF ULTRASOUND-GUIDED FOAM SCLEROTHERAPY COMBINED WITH SAPHENO-FEMORAL LIGATION AND SURGICAL TREATMENT OF VARICOSE VEINS"** has been done by me. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other University board either in India or abroad.

This is submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of Master of Surgery degree Branch –I(General surgery) to be held in March 2009.

Place : Madurai **DR PRATHBA.N**

Date :

ACKNOWLEDGEMENT

I am greatly indebted to **Dr.M. GOBINATH, M.S ,** Professor and Head of the Department of General surgery, Madurai Medical College, Madurai for his guidance and encouragement in preparing this dissertation.

My heartful thanks to **Prof.Dr. RAJAGOPALAN, M.S, MCh,** Chief of vascular surgery , Madurai Medical College, Madurai for his guidance in doing this work.

My sincere thanks to **Dr R.Vijayan, M.S, FICS.,** Additional Professor of Surgery, Madurai Medical College, Madurai for his able assistance in completing this study.

I also thank my Assisstant Professors **Dr.P. Sundareshwari M.S, DGO, Dr. Lakshmi Balan M.S, DGO, Dr. M.Muthukumar, M.S** and **Dr.P. Prabakaran, M.S** for their constant support and guidance in performing this study.

I also thank my post graduate colleagues, for their kind co-operation in doing this study.

My profound thanks to **Dr.S.M. Sivakumar, M.S., Dean**, Madurai Medical College and Government Rajaji Hospital, Madurai for permitting to utilize the clinical materials of this hospital in the completion of my dissertation. I gratefully acknowledge the patients who give their consent and co-operation for this study

CONTENTS

INTRODUCTION

Varicose veins affect 20–30% of adults. People with venous insufficiency maysuffer from heaviness, aching, throbbing, itching and cramps or fatigue in the legs. In some patients, chronic venous insufficiency can lead to skin discoloration,inflammatory dermatitis and skin ulceration.

Conservative methods such as compression hosiery may improve symptomatic varicose veins. If symptoms persist, the main treatment options are sclerotherapy,surgery (usually stripping and ligation of the long or short saphenous veins and phlebectomies), and ablation (by laser or radiofrequency ablation).

Treatment of venous insufficiency with liquid sclerotherapy is considered by some to be an unfulfilled promise . It was heralded in the first half of the last century to be a replacement for surgery but as recurrences of varicose veins appeared in limbs treated with injection techniques, surgery re-appeared and was dominant in the last half of the century . Administration of foamed sclerosant was reintroduced in the early 1990s by Cabrerra, who summarized a broad experience in 1997 .

Foam in sclerotherapy must be looked upon as an entirely new method of treatment. Ultrasound-guided foam sclerotherapy for varicose veins is a variation of liquid sclerotherapy in which sclerosant foam is injected into the affected veins using ultrasound monitoring. The foam causes inflammation of the vein wall, leading to obliteration and occlusion of its lumen.

It is useful in all types of varices and it is proven to be safe, simple, cheap, reliable and repeatable. it is a potentially useful treatment for both main trunk and minor vein disease, and can be conducted as an outpatient procedure without the requirement for general or regional anaesthesia. This could have implications in terms of releasing theatre time and space for other surgical procedures. In addition, foam sclerotherapy can be delivered in an outpatient setting, and possibly also a primary care setting, as long as the surgeon is appropriately trained and adequate diagnostic and monitoring facilities are available.

This work consists of a detailed study of 50 patients with varicose veins who were admitted in Govt. Rajaji Hospital , Madurai, between 2006 and 2007.

AIM OF THE STUDY

1. To compare the efficacy of treatment of varicose veins with Saphenofemoral ligation and Foam sclerotherapy compared to Saphenofemoral ligation and stripping.

2. To analyze the merits and demerits of Foam sclerotherapy.

3. To study the complications following Foam sclerotherapy.

ANATOMY

The peripheral venous system functions both as a reservoir to hold extra blood and as a conduit to return blood from the periphery to the heart and lungs. Unlike arteries, which possess three well-defined layers, most veins are composed of a single tissue layer. Only the largest veins possess internal elastic membranes, and at best this layer is thin and unevenly distributed, providing little buttress against high internal pressures. The correct functioning of the venous system depends on a complex series of valves and pumps that are individually frail and prone to malfunction.

The Superficial Venous System

The superficial venous system is extremely variable weblike network of interconnecting veins, most of which are unnamed. A few larger truncal superficial veins are fairly constant in location; These truncal superficial veins serve as a conduit to pass blood centrally and eventually into the deep venous system.

The principal named superficial veins of the lower extremity are the short saphenous vein (SSV), which usually runs from ankle to knee, and the greater saphenous vein (GSV), which usually runs from ankle to groin.

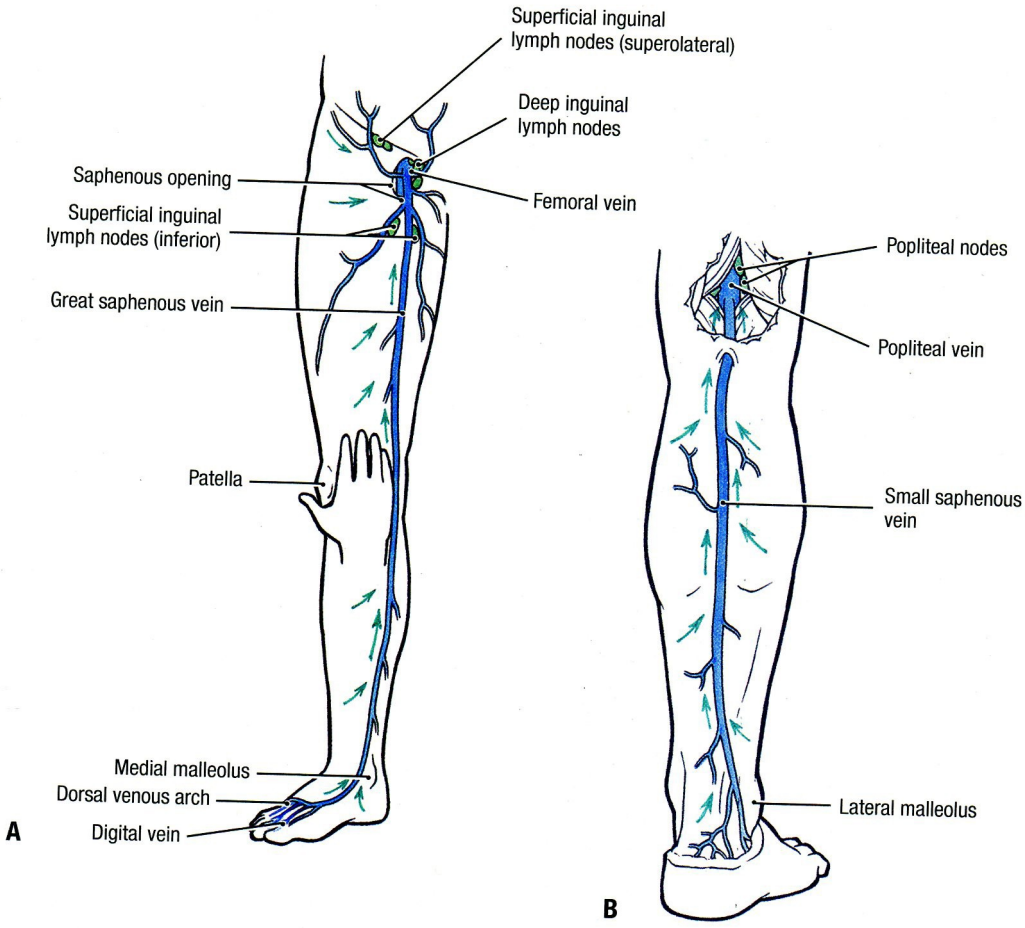
Anatomy of the Short Saphenous Vein

The short saphenous vein originates in the lateral foot. It passes posteriorly lateral to the Achilles tendon in the lower calf. The SSV usually lies directly above the deep fascia in the midline as it reaches the upper calf, where it enters the popliteal space between the two heads of the gastrocnemius muscles. In two thirds of cases, it joins the deep popliteal vein above the knee joint, and in one third of cases, it joins with other veins (most often the GSV or the deep muscular veins of the thigh).

Anatomy of the Greater Saphenous Vein

The GSV originates in the medial foot and passes upward anterior to the medial malleolus, then crosses the medial tibia in a posterior direction to ascend in the medial line across the knee. Above the knee it continues anteromedially above the deep fascia to the thigh, where it passes through the foramen ovale and joins the (deep) common femoral vein at the groin crease.

SUPERFICIAL VENOUS SYSTEM



Tributaries at the Sapheno femoral junction

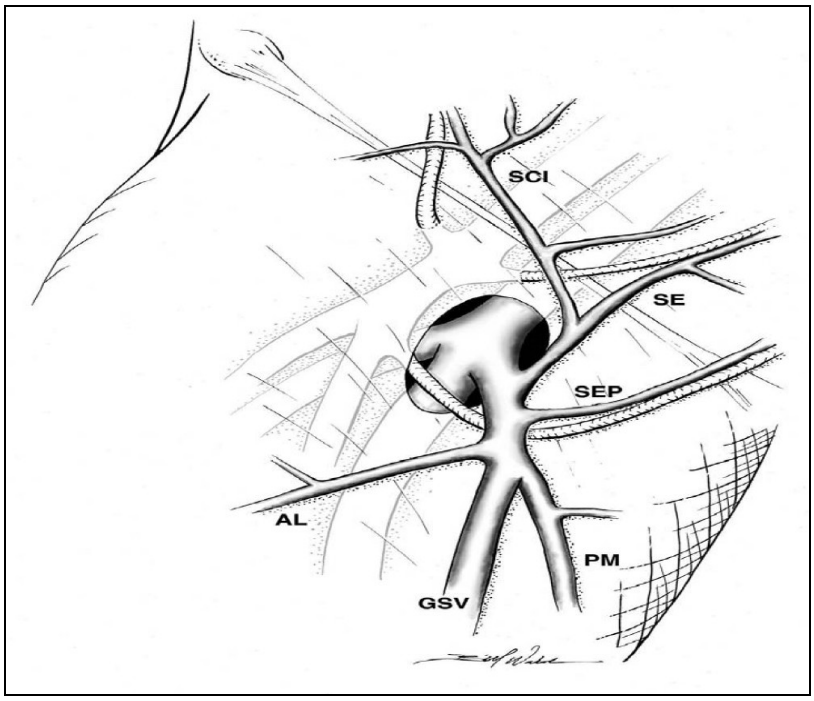
1. AL- Anterolateral

2. PM- Posteromedial

3. SEP- Superficial external pudendal

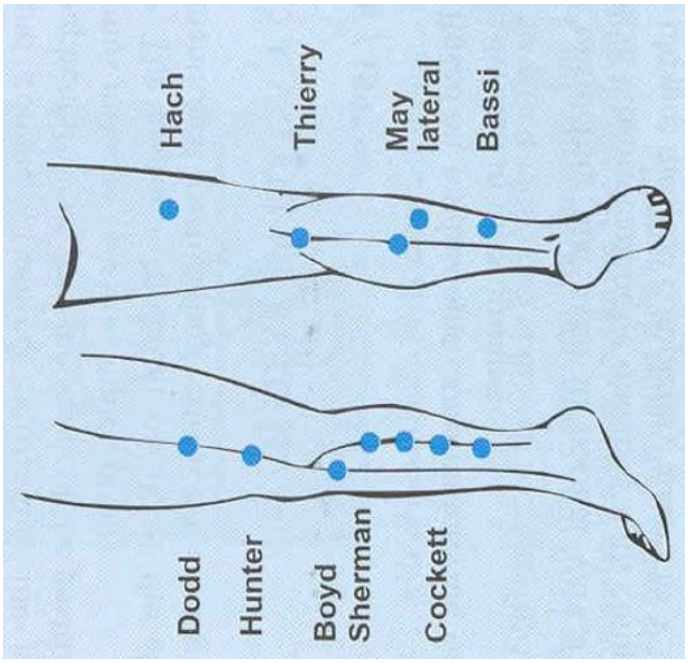
4. SCI- superficial circumflex iliac

5. SE – Superficial epigastric



Perforating Vein

The SPJ and SFJ are not the only pathways from the superficial system the deep system. Both the superficial collecting web and the superfici truncal veins are also connected to a variable number of perforating veins that pass through anatomic defects in the deep fascia to join directly with the deep veins of the calf or thigh. Perforating veins usually contain venous valves that prevent reflux of blood from the deep veins into the superficial system. A few named perforating veins are fairly constant in location and are named only as vague groupings.



The Deep Venous System

All venous blood eventually is received by the deep venous system on its way back to the right atrium of the heart. In most cases there are five major named branches to the deep venous system, three below and two above the knee.

Deep Veins of the Calf

In the lower leg, three groups of deep vein exist: the anterior tibial vein (ATV), draining the dorsum of the foot; the posterior tibial vein (PTV), draining the lateral aspect of the foot. From the ankle, the anterior tibial vein passes upward anterolateral to the interosseous

membrane, the posterior tibial vein passes upward posteromedially beneath the medial edge of the tibia, and the peroneal vein passes upward posteriorly through the calf. Venous sinusoids within the calf muscle coalesce to form soleal and gastrocnemius intramuscular venous plexi, which join the peroneal vein in midcalf. In most patients, each one of these is actually a pair of veins flanking an artery of the same name; thus there are actually six named deep veins below the knee in a typical patient. Just below the knee, the four anterior and posterior tibial veins join with the two peroneal veins to become the single large popliteal vein

Deep Veins of the Thigh

The popliteal vein courses proximally behind the knee and then passes anteromedially in the distal thigh through the adductor canal, at which point it is called the FV.The common femoral vein (CFV) passes upward above the groin crease to become the iliac vein.

The Calf Muscle Pump .

The passage of blood upward from the feet against gravity depends on a complex array of valves and pumps. Muscle pumps of the calf and thigh provide the motive force for venous return. The most important of

these is called the calf muscle pump, often referred to as the peripheral heart. Each segment of the calf muscle pump works in the same way as the hand bulb of the sphygmomanometer. Inflow to a segment of deep vein is through intake valves from perforating veins as well as from the deep vein segment below. Outflow is through an outflow valve to the deep vein segment above. Squeezing of the vein segment occurs when muscle contraction increases the pressure within a fascial muscle compartment.

Pathophysiology

Venous pathology develops when venous return is impaired for any reason. Venous pathology can be deep, superficial, or mixed. It can result from primary muscle pump failure, from venous obstruction (thrombotic or nonthrombotic), or from venous valvular incompetence, which may be segmental or whole leg When the entire venous system is filled, the valves float open and venous pressure rises to a maximum exactly equal to the height of the standing column of venous blood from head to foot. This condition triggers an urge to move the legs, activating the muscle pumps and re-emptying the legs.

Deep Vein Obstruction

Partial obstruction of the deep veins may have little effect on venous outflow, but severe obstruction of the deep veins produces

secondary muscle pump failure. In this case the muscle pump produces an appropriately high outflow pressure with each contraction, but the volume of venous blood pumped out of the calf is reduced because of the reduced diameter of the outflow tract.

Deep Vein Incompetence

If outflow tracts are open and the muscle pump is functional but the valves of the deep veins permit reflux (because of primary agenesis, prior thrombosis, direct trauma, or dilatation with secondary valvular failure), venous blood will be pumped out of the calf in normal volumes but extremity refill will include both normal arterial inflow and pathologic venous retrograde flow. The venous pressure immediately after ambulation may be slightly elevated or it may even be normal, but the veins will refill and dilate very quickly. After a person with deep incompetence stands for only a few seconds, the venous pressure will be nearly as high as the maximum reached after prolonged standing. Again, the volume of venous blood that suffuses the extremity and dilutes arterial inflow will be somewhat increased. A smaller fraction of the extremity's venous blood will return to the central circulation each minute, and because arterial blood must flow into congested tissues with elevated hydrostatic pressure, the volume of arterial inflow will again be somewhat reduced.

Perforator Incompetence

Under ordinary circumstances the bulk of venous blood moves strictly from the superficial to the deep system. Failure of the valves of communicating perforator veins can permit a significant volume of blood to flow from deep veins backward into the superficial system, producing local congestion and venous hypertension. More important, perforator incompetence allows the extremely high pressures generated within deep veins by the calf muscle pump to be communicated to the superficial veins, which are not strong enough to tolerate the pressure. This high pressure (even if intermittent and highly localized) can produce excessive venous dilatation and secondary failure of superficial vein valves. This is one of the major mechanisms for the development of superficial venous incompetence and varicose veins.

Superficial Incompetence

Superficial venous incompetence is the most common form of venous disease. Retrograde flow through the superficial venous system occurs when venous valve no longer perform their usual function. This can happen for a variety of reasons. Direct injury or superficial phlebitis may cause primary valve failure. In most cases, however, superficial venous reflux is simply the inevitable end result of the introduction of high pressures into otherwise normal superficial veins that were intended

to function as a low- pressure system. High pressure causes normal superficial veins to dilate so widely that the thin flaps of venous valves simply no longer meet in the midline.

High pressure can enter the superficial veins by failure of key valves at any point of communication between the deep and superficial systems. Two clinical syndromes of high pressure superficial system disease are recognized: junctional and perforator. Junctional high-pressure disease results from failure of the primary valve at the junction between the GSV and the CFV (the SFJ), or at the junction between the SSV and PV (the SPJ). Vein dilatation in these cases proceeds from proximal to distal, and patients perceive that a large vein is "growing down to the leg." Perforator high-pressure disease results from failure of the valves of any perforating vein. The most common sites of primary perforator valve failure are at the canal of Hunter in the midproximal thigh (Hunterian vein) and in the proximal calf. If the primary high-pressure entry point is distal, patients experience the initial development of large clusters of veins in the lower leg, with large veins eventually "growing up the leg" toward the groin.

ANATOMY		
Superficial venous system	**Perforator system of communicating veins**	**Deep venous system**
long saphenous vein: • runs from the medial side of the dorsal venous arch up the anteromedial aspect of the leg and thigh until it empties into the femoral vein	• these veins pass through the deep fascia and carry blood from the superficial venous system to the deep venous system, one way flow maintained by valves • however these valves may become	• comprises • femoral and popliteal veins • venae comitantes ie veins accompanying the ant. tibial, post. tibial and peroneal arteries • valveless blood lakes within calf muscle • communicates with superficial system via

short saphenous vein • runs from the lateral side of the dorsal venous arch up the posterolateral aspect of the calf until it passes through the popliteal fascia behind the knee and empties into the popliteal vein just above the knee	incompetent and cause appearance of superficial varicose veins during exercise	a. saphenofemoral junction b. mid-thigh perforators c. short sapheno-popliteal junction d. calf perforators

PATHOPHYSIOLOGY AND CLINICAL FEATURES

There is a familial tendency toward development of varicose veins. In clinical practice, usually 70% of patients can identify superficial venous disease as a familial trait. In addition, the severity of venous disease may also be familial. Patients whose parents had venous ulceration are at increased risk of this complication as well.

Female hormonal influences on the veins are profound. For example, when Varicose veins develop in pregnancy, in 70-80% of cases, they appear in the first trimester. This development is not dependent upon size of the gravid uterus, increased blood flow, or iliac venous occlusion, but is due to the immediate rise of progesterone. It has

been found that diameter of the femoral vein increases by 50% within six weeks of the start of pregnancy.

Two forces act on the hereditary/hormonal substrate to produce dilation and elongation of veins. The first, the weight of the blood column from the right atrium to the affected veins is a constant gravitational force and may be referred to as hydrostatic pressure. This is measured in millimeters of water. The Basle study demonstrated that among workers in a factory, those with standing jobs were significantly more likely to have venous disease, followed by those with sitting jobs. The adoption of a regular exercise program is followed by a reduction in the symptoms of aching and pain. The second is pressure exerted by contracting muscles on adjacent veins. This may. be called a hydrodynamic force. This pressure, measured in millimeters of mercury (13 times heavier than water) propels blood through open deep valves toward the heart. However, when valves in perforating veins fail, this force is exerted on subcutaneous veins and venules in the epidermis in a pulsatile fashion coincident with muscular contraction. It is no wonder that they become elongated and tortuous and that venous dilation causes their valves to fail in turn. In summary, it follows that telangiectasias, reticular varicosities, and varicose veins are derived from a hereditary/hormonal substrate which is acted upon by static gravitational pressure and dynamic

muscular forces transmitted through failed venous valves. While neither the hereditary nor the hormonal substrate can be altered by intervention, the influence of hemodynamic forces and hydrostatic pressure can be modified by intervention.

Chronic venous insufficiency/venous ulcers

CVI is manifested by lower limb oedema and lipodermatosclerosis, i.e. skin changes such as pigmentation, atrophy and eczema. It arises secondary to venous hypertension. The high pressure leads to oedema and leakage of protein has local inflammatory effects.

A chronic venous ulcer can be defined as an area of discontinuity of epidermis, persisting for four weeks or more and occurring as a result of venous hypertension and calf muscle pump insufficiency. It is easily recognised when it is situated in the 'gaiter' region near the medial malleolus (the protuberance at the lower end of the tibia), and occasionally adjacent to the lateral malleolus (the protuberance at the lower end of the fibula); it has a shallow base with a flat margin and the surrounding skin has features of long standing venous hypertension, i.e. haemosiderin pigmentation, atrophie blanche, eczema, and dilated venules over the instep of the foot.

Venous ulcers are located at the severe end of the spectrum of chronic venous disorders of the leg . Because of this they occur more often in subjects with other forms of venous diseases such as varicose veins and skin changes. Venous leg ulceration has been reported to account for 70-95% of all leg ulcers and 20-50% of these are said to be a consequence of varicose veins. However, there is a general lack of data explaining the way varicose veins develop into venous ulcers, and at what point treatment could be advised as being prophylactic rather than remedial.

Venous hypertension is the undisputed initiating factor in venous ulcer development. More severe venous incompetence is associated with a higher risk of ulceration. The higher the venous pressure, the greater the risk, whether incompetence involves the deep or superficial venous system. The reasons why venous ulcers go on to develop is still an area of debate but there are three main theories .

An important determinant of venous ulcers is the venous hypertension that can arise after deep vein thrombosis, especially if it extends above the knee. Brandjes *et al* have shown that wearing a compression stocking can reduce the incidence of the post-thrombotic limb. This condition commonly occurs after DVT; in the Brandjes study of DVT it occurred in 60% of the control arm within 2 years.

Events which cause venous ulceration - three theories

- **Fibrin cuff:** Excessive venous pressure causes large molecules of fibrinogen to leak out into superficial tissues because capillary walls are only one epithelial cell thick. Fibrinogen is then polymerised into fibrin cuffs around capillaries, which prevents oxygen and nutrients from diffusing out from the capillaries, leading to cell death and ulceration.
- **White cell trapping:** Venous hypertension causes white blood cells to become trapped and accumulate in the capillaries of dependent legs (legs which are allowed to hang down in the dependent position for long periods). This leads to capillary occlusion, the release of protolyctic enzymes and toxic metabolites, which result in local ischaemia and ulceration.
- **A combination of mechanisms:** A cascade of events is initiated by venous hypertension. White blood cells release cytokines which stimulate other cells to synthesise fibrin cuffs. These cuffs inhibit the development of new blood vessels and deprive superficial tissues of oxygen and nutrients, leading to tissue damage and ulceration.

Risk factors

Risk factors for varicose veins include fixed factors - female sex, age, pregnancy, ethnicity, geographic location, left iliac vein compression

by the right iliac artery, family history - and potentially preventable factors - obesity, occupations requiring prolonged standing or sitting, lack of dietary fibre, use of constricting corsets and sitting posture for defecation

Sex

It is generally believed that women are more commonly affected by varicose veins than men and most studies have shown a female predominance of varicose veins.

In the majority of studies the sex ratio decreases with increasing age.

Age

The association between age and prevalence of varicose veins is fairly conclusive. There is a steady increase in prevalence of varicose veins with increasing age for all grades of varicosity. The same trend was found in the prevalence of reticular and hyphenweb varices

Pregnancy

It is generally believed that pregnancy leads to varicose veins due to the pressure of the uterus obstructing venous return from the legs. However, this has been refuted, as the majority of varices appear during the initial 3 months when the uterus is not large enough. A hormonal factor is thought to be responsible or the increased circulating volume of blood.

Family history

A number of studies have found that the risk of varicose veins was higher in those with affected relatives, perhaps suggesting a genetic element or shared environmental factors. There is often a clear family history of the disorder with some patients inheriting abnormalities in the FOXC2 gene.

Body weight and height

Several studies have found an association between weight and body mass and an increased risk for varicose veins. there was an increased prevalence of varicose veins with increasing body weight

Occupation

A person's occupation is a possible risk factor for varicose veins. A standing occupation has been indicated in some studies as a significant risk factor for varicose veins

OTHER RISK FACTORS

1. Smoking
2. Constricting undergarments
3. Post- thrombotic limb
4. Constipation

A diet deficient in fibre has been implicated as a major factor in the causation of varicose veins. It is thought that fibre-depleted diets lead to constipation and the subsequent straining to produce a stool produces high intra-abdominal pressures which are transmitted to the leg veins and progressively dilate them.

VARICOSITY OF GSV

HEALED VENOUS ULCER

CLINICAL FEATURES :

- Asymptomatic
- aching in the calf particularly towards the end of the day
- pigmentation
- ankle swelling
- itching
- eczema
- venous ulceration

CLASSIFICATION AND GRADING

CEAP classification

Limbs with chronic venous disease are classified according to clinical signs (C), cause (E), anatomic distribution (A), and pathophysiologic condition (P)

Clinical classification

C0 : no visible or palpable signs of venous disease

C1 : telangiectasies or reticular veins

C2 : varicose veins

C3 : edema

C4a : pigmentation or eczema

C4b : lipodermatosclerosis or atrophie blanche

C5 : healed venous ulcer

C6 : active venous ulcer

S : symptomatic, including ache, pain, tightness, skin irritation, heaviness, and muscle cramps, and other complaints attributable to venous dysfunction

A : asymptomatic

Etiologic classification

Ec: congenital

Ep: primary

Es: secondary (postthrombotic)

En: no venous cause identified

Anatomic classification

As: superficial veins

Ap: perforator veins

Ad: deep veins

An: no venous location identified

Pathophysiologic classification

Basic CEAP

Pr: reflux

Po: obstruction

Pr,o: reflux and obstruction

Pn: no venous pathophysiology identifiable

Venous Clinical Severity Score (VCSS)

Attribute	**Absent = 0**	**Mild = 1**	**Moderate = 2**	**Severe = 3**
Pain	None	Occasional	Daily	Limit activities
Varicose veins	None	Few, scattered	Multiple (LSV)	Extensive (LSV, SSV)
Venous edema	None	Evening, ankle	Afternoon, leg	Morning, leg
Pigmentation	None	Limited area	Wide (lower 1/3)	Wider (above 1/3)
Inflammation	None	Cellulitis	Cellulitis	Cellulitis
Induration	None	Focal (< 5 cm)	< lower 1/3	Entire lower 1/3
Number of AC	0	1	2	3
Duration of AC	None	< 3 months	3 months – 1 year	> 1 year
Size of AC	None	< 2 cm diameter	2-6 cm diameter	> 6 cm diameter
Comp therapy	Not used	Intermittent use	Most days	Continually
LSV, long saphenous vein; SSV, short saphenous vein; AC, active ulceration; lower 1/3, lower 1/3 of the leg.				

Venous Disability score (VDS)

Score	Definition
0	Asymptomatic
1	Symptomatic, but able to carry out usual activities* with-out compressive therapy
2	Able to carry out usual activities* only with compression and/or limb elevation
3	Unable to carry out usual activities* even with compression and/or limb elevation
*Usual activities = patients activities before the onset of disability due to venous disease.	

MANAGEMENT

INVESTIGATIONS

Standard Doppler examination

A standard Doppler probe emits a sound when blood flows past the transmitting and receiving crystals. A uniphasic signal indicates flow in one direction. A biphasic signal indicates forward and reverse flowand is indicative of blood refluxing down through incompetent valves.A Doppler probe is placed over the Saphenofemoral junction.A calf squeeze is carried out and if a biphasic signal is obtained this confirms the presence of incompetence of saphenofemoral junction.This is not an accurate method for establishing incompetence of the lesser saphenous vein as its termination is variable and it is difficult to separate lesser saphenous incompetence from popliteal valvular incompetence. In all cases of short saphenous incompetence further investigation is desirable; this is usually carried out by duplex scanning

Duplex ultrasound imaging

The probe of a dulplex scanner contains multiple emitting and receiving crystals. These allow a grey scale image to be obtained in which the veins are seen as a black void in the subcutaneous and deep tissues. Directional flow can be shown as a colour image (red or blue) superimposed on the grey scale image of the vessel. Visible venous flow can only be seen when augmented by a calf squeeze. The B-mode grey-scale image allows the vein to be traced to its termination,while

compression and relaxation , or a Valsalva manoeuvre, may demonstrate the presence of retrograde flow.

Varicography

In this investigation contrast is injected directly into surface varices. The contrast is non-thrombogenic, as it is non- ionic and iso-osmolar with blood. This allows detailed mapping of the varices to their termination.This is an extremely useul investigation in patients with recurrent varicose veins or those with complex anatomy.

Venography

In this investigation tourniquets are used to direct contrast injected into the superficial veins of the foot into the deep veins of the calf, thigh and pelvis. It is not used as a standard investigation in patients with varicose veins but is useful if the Duplex scan indicates but cannot confirm, the presence of post thrombotic change.

TREATMENT

Current treatment options for venous disease include compression hosiery, Sclerotherapy (liquid or foam) with or without ultrasound guidance, endovenous laser ablation treatment (EVLT), radiofrequency ablation, open surgery (usually sapheno-femoral ligation, stripping of the long saphenous vein and phlebectomies or sapheno-popliteal ligation, and phlebectomies), and subfascial endoscopic perforator surgery alone or in combination.

Indications for interventions include recurrent episodes of haemorrhage and the development of skin changes(lipodermatosclerosis or ulceration) associated with an ankle flare indicative of venous hypertension.

Patients should be treated with elastic compression stockings if the varicose veins are found to be associated with post thrombotic damage.Most patients are prescribed class II stockings with an ankle pressure of around 30mmHg decreasing to 10-15mmHg the knee level. Above – knee stocking should never be prescribed.Class I stockings can be used to control simple varicose veins.

Sclerotherapy

During the last half of the 20th century, sclerotherapy as a major treatment of varicose veins came and went. At first, it was widely heralded as a substitute for surgery but after a prospective randomized study by Hobbs, interest in sclerotherapy waned. In this technique a detergent is injected directly into the superficial veins. The detergent destroys lipid membranes of endothelial cells causing them to shed leading to thrombosis, fibrosis and obliteration(sclerosis).

Fegan stressed the importance of continued local compression following sclerosant injections to reduce the incidence and amount of superficial thrombosis and improve the`sclerosis` of the vein .

Foam sclerotherapy

Just before the turn of the 21st century, Cabrera published his experience with foamed sclerosant in patients with great saphenous varices and arteriovenous malformations. Cabrera designed his treatment with the specific aim of obliterating the saphenous trunks. His technique consisted of filling the great saphenous vein in the thigh or the small saphenous vein in the calf with foamed sclerosant injected under ultrasound control. His initial report on long-term follow-up revealed that the results were at least comparable to surgery and perhaps somewhat better and his results have been confirmed by others. Investigations into treatment of small vein varices, including telangiectasias, has resulted in

the finding that foam results in a 20% improved appearance compared to liquid sclerosant. The most popular sclerosants currently used as foams are polidocanol and sodium tetradecyl sulfate and of the many techniques used in making foams, the technique of Tessari has proven most popular

Foam sclerotherapy is a variant of sclerotherapy. It differs from liquid sclerotherapy in that instead of injecting a liquid sclerosant solution into the vein, the sclerosant is transformed into foam by forcibly mixing it with air or other type of gas.

Tessari developed an easy way of making liquid sclerosant into foam using two syringes and a three-way stopcock

The foam solution has the consistency of shaving cream, which improves treatment in two distinct ways. First, the foam displaces blood within the vein, permitting the full strength of the sclerosing agent to be in direct contact with the vein wall for an extended period of time without any dilution effects. Second, the foam is visible via ultrasound imaging and can be easily tracked and guided to the source of the venous problem The question arises of why foam sclerosant treatment of CVI succeeds when liquid sclerotherapy treatment of CVI has made little impact. Foam improves the contact with the endothelium of the vein compared to liquid sclerosants with an improved effect .The endpoint of

TESSARI TECHNIQUE OF PRODUCING FOAM

successful sclerotherapy is irreversible venous vascular fibrosis. This only occurs in response to endothelial cell destruction with exposure of the subendothelial layer of cells. This is accomplished by detergent sclerosants which work by the mechanism of protein theft denaturation in which an aggregate of detergent molecules forms a lipid bilayer in the form of a cylinder, a sheet or micelle. This disrupts the endothelial cell surface by stealing away the essential proteins from the cell membrane surface and producing delayed cell death.

Potential adverse events associated with sclerotherapy (liquid and foam) include allergy, thrombophlebitis, pigmentation, nerve damage, cutaneous necrosis, ulceration and matting. There are concerns that the sclerosant can potentially enter the deep veins and induce venous thromboembolism. There are additional concerns that, particularly in people with a Patent Foramen Ovale (PFO), the sclerosant may enter the arterial circulation and induce an ischaemic stroke or retinal artery occlusion. The use of foam may increase this risk. Transient visual disturbances are reported following sclerotherapy. The mechanism is uncertain but is felt to be vasospastic, and similarly the risk may be higher in foam compared with liquid sclerotherapy.

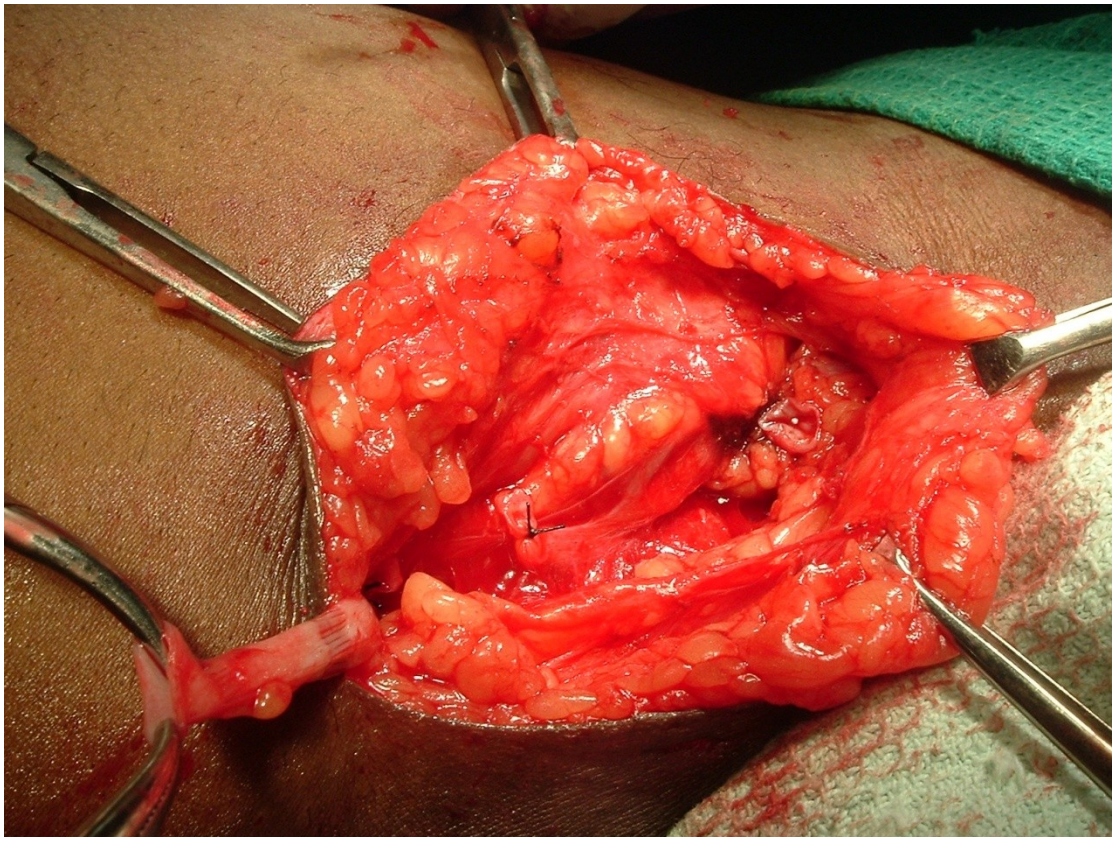
Saphenofemoral junction ligation

An oblique incision is made in the groin centered 2.5 cm below and lateral to pubic tubercle**.** The greater saphenous vein is found in the superficial fat by blunt dissection and traced upwards to its `T` shaped termination with the Femoral vein. This must be clearly established before the vein is divided.As the vein is traced to its termination four tributaries are normally encountered. These are the superficial inferior epigastric vein,the superficial circumflex iliac vein and the deep and superficial external pudendal veins.The long saphenous veinis not ligateduntil the T-junction has been confirmed and the femoral vein has been exposed for a centimeter in either direction

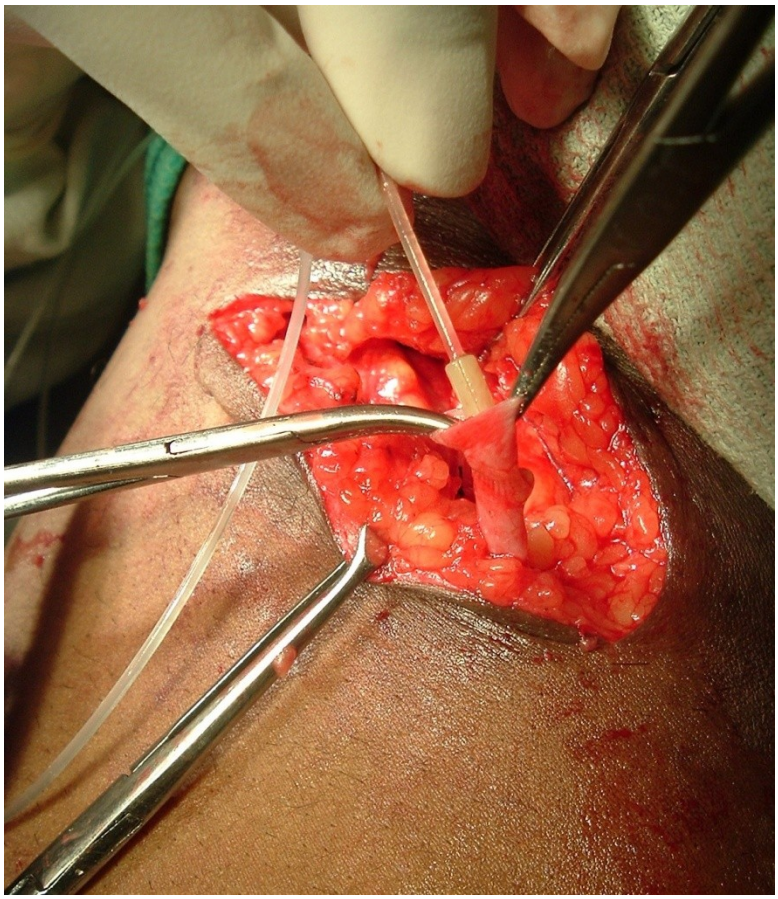
Greater saphenenous stripping

The saphenous trunk is retrogradely stripped to the knee. Either a variation of Babcock`s intraluminal stripper or a rigid metal `pin` stripper is used which invaginates the vein and causes less bruising. Stripping not only avulses the main vein trunk but also pulls out the termination of the tributaries

SAPHENOFEMORAL JUNCTION LIGATION



GSV STRIPPING



Radiofrequency ablation

In this technique a catheter is passed up the saphenous vein from the lower leg and withdrawn under ultrasound control while radiofrequency waves are used to destroy the endothelial lining through a series of metal prongs.

Endovenous laser ablation

It is a more recent development. A laser probe is passed up inside a catheter inserted into the lowerpart of the saphenous vein under ultrasound guidance. It is important that large amounts of crystalline fluid containing local anaestheic are placed around the vein to seperate the skin from the laser probe and avoid cutaneous burns. The laser probe is withdrawn from the sapheno femoral junction , administering a set number of joules to the endothelial lining of the saphenous vein. The laser probes are marginally less expensive than radiofrequency ablation.

Both laser and RFA do not provide `flush` occlusion of the saphenous vein, often leaving tributaries that would always be divided at surgery. Long term studies are required to ascertain the recurrence rate of both techniques.

Subfascial endoscopic perforator vein surgery

Preoperative evaluation is performed by duplex scanning of the superficial, deep and perforator venous systems to diagnose both valvular incompetence and obstruction. Standard laparoscopic equipment is used. At operation the limb is exsanguinated, and two endoscopic ports (typically a 10 mm and a 5 mm port) are placed in the subfascial space in the calf at two sites remote from the area of venous ulceration. A space-maker balloon is introduced and inflated in this subfascial space to improve access. Carbon dioxide is then insufflated to facilitate dissection. The incompetent perforating veins are clipped and divided using endoscopic scissors or alternatively, coagulated and divided using an ultrasonic coagulator (harmonic scalpel).

REVIEW OF LITERATURE

1. Bountouroglou DG et al (Eur J Vasc Endovasc Surg, Jan 2006) Conducted a prospective randomised controlled trial comparing sapheno-femoral ligation, great saphenous stripping and multiple avulsions with sapheno-femoral ligation and ultrasound guided foam sclerotherapy to the saphenous vein. Primary end points were patient recovery period and quality of life and secondary end points frequency of complications on the two arms of the trial and the cost of the treatment. Sixty patients were included in the trial. They concluded that Ultrasound guided sclerotherapy combined with sapheno-femoral ligation was less expensive, involved a shorter treatment time and resulted in more rapid recovery compared to sapheno-femoral ligation, saphenous stripping and phlebectomies.

2. Rabe .E, Otto. J(Eur J Vasc Endovasc Surg.feb 2008) Compared standardised polidocanol foam to liquid polidocanol in a randomized controlled trial. 106 patients with primary varicose veins due to an incompetent GSV were treated with either standardised 3% polidocanol foam or 3% liquid polidocanol. The primary efficacy criterion was elimination of reflux (<0.5 sec) measured

3. cm below the sapheno-femoral junction (SFJ) by duplex ultrasonography 3 months after the last injection. The results showed that a significantly greater number of patients were successfully treated by foam sclerotherapy resulting in 69% elimination of reflux compared to 27% patients treated with liquid sclerosant Abela.R et al(Eur J Vasc Endovasc surg . Oct 2008) Compared Reverse Foam Sclerotherapy of the great saphenous vein (GSV) combined with sapheno-femoral junction (SFJ) ligation to standard (Babcock) stripping and invagination (Pin) stripping in a prospective clinical series. They found that standard stripping of the GSV and invagination stripping are not associated with major discomfort and problems in the early post-operative period. SFJ ligation and GSV reverse foam sclerotherapy yielded greater patient satisfaction with less post-op bruising and discomfort and reduced analgesic requirements

4. Rutgers. PH, Kitslaar. PJ(Am J Surg Oct 1994) compared the treatment of greater saphenous vein insufficiency by stripping and local avulsions of varicose veins with high ligation of the saphenofemoral junction (crossectomy) combined with sclerocompression therapy. Of 156 consecutive patients, 89 legs were randomly allocated to stripping and 92 to high ligation. At

follow-up of 3 months and 1, 2, and 3 years after treatment, clinical and Doppler ultrasound results, and complaints and cosmetic results, as judged by the patient and the surgeon, were scored. The results of treatment of isolated saphenous vein insufficiency by stripping operation, therefore, were superior to those obtained by high ligation combined with sclerotherapy.

5. Miyazaki K et al (Int Angiol Sep 2005) Determined the long-term recurrence rates of greater saphenous vein (GSV) insufficiency after treatments for primary varicose veins. The recurrence - free rates after stripping surgery, saphenofemoral ligation, and sclerotherapy were estimated. The saphenofemoral ligation group and sclerotherapy group had significantly higher recurrence rates than the stripping group. There was no difference in recurrence rates between the saphenofemoral ligation group and sclerotherapy group

6. Yamaki T, Nozaki M (Eur J Vasc Endovasc surg Oct 200 Compared the proportion of foam sclerosant that enters deep veins between multiple injections of <0.5ml foam per injection and a few injections of >0.5ml foam per injection. One hundred and seven patients with superficial venous incompetence were randomised to

receive either multiple injections of <0.5ml 1% polidocanol (POL) foam (multiple injections) or a few injections of >0.5ml 1% POL foam per injection (few injections) for the treatment of varicose tributaries. These findings suggest that multiple small-dose injections can reduce the amount of foam sclerosant and the risk of foam sclerosant entering the deep veins in patients with superficial venous insufficiency.

MATERIALS AND METHODS

This is a prospective randomized study conducted at Government Rajaji Hospital, attached to Madurai Medical College, Madurai, from the year 2006-2007.

After obtaining approval by the ethics committee and informed consent, a total of 50 patients of either gender with varicose veins of the lower limbs , including those with venous ulcers were enrolled for this study. Patients with recurrent varicose veins , multiple perforator incompetence, allergy to sclerosant and history of deep vein thrombosis were excluded from the study.

All patients underwent Doppler scanning to identify Saphenofemoral incompetence, the sites of incompetent perforators and to rule out deep vein thrombosis. The patients were randomly allocated to one of the two groups

Group 1 : SF ligation with foam sclerotherapy

Group 2 : SF ligation and stripping

Sixty patients with primary varicose veins due to GSV incompetence and suitable for day care surgery were randomly allocated to undergo ultrasound-guided sclerotherapy with sapheno-femoral ligation under local anaesthesia (n=25) or sapheno-femoral ligation, stripping and multiple avulsions under general or regional anaesthesia (n=25). The study protocol included history, physical examination, assignment of CEAP class and assessment venous clinical severity score (VCSS), and colour duplex ultrasound to identify sites of incompetent perforators and to rule out deep vein thrombosis.

PREPARATION OF FOAM:

The Tessari technique was used to convert liquid sclerosant (3% sodium tetradecyl sulphate) to foam. Two 5cc syringes are connected by a three-way valve and the fluid sclerosant is forcibly mixed with air at a ratio of liquid: air of 1:4 and frothed into foam by a pumping action.

TECHNIQUE OF SCLEROTHERAPY:

Sclerotherapy was conducted as an outpatient procedure without anaesthesia.The vein was cannulated at the ankle with a scalp vein needle and foam injected.

Duplex ultrasound imaging was used to guide cannulation, monitor the injection and flow of foam and to minimise the risk of foam

diffusion to the deep venous system. More than one injection was used during the same session in an attempt to ensure that all of the target veins have been completely filled. Localised compression with the ultrasound probe was applied over the main sites of communication between the superficial and deep venous systems i.e sapheno-femoral, sapheno-popliteal junction,and perforators when truncal veins were injected, with the aim of avoiding entry of sclerosant into the deep veins.

The limb was held elevated at an angle of 45º and compression at the sites of communication applied for a period of 5 min. Elastocrepe bandages were applied to maintain compression and patient was advised strict bed rest for a period of 6 hrs. A maximum of 10ml of foam was used in a session. More than one treatment session was used in case of extensive varicosities.

Sapheno-femoral ligation was done under local anaesthesia after a period of 2 days.

Patients in the control group underwent Sapheno- femoral ligation , stripping and multiple avulsions under general or regional anaesthesia.

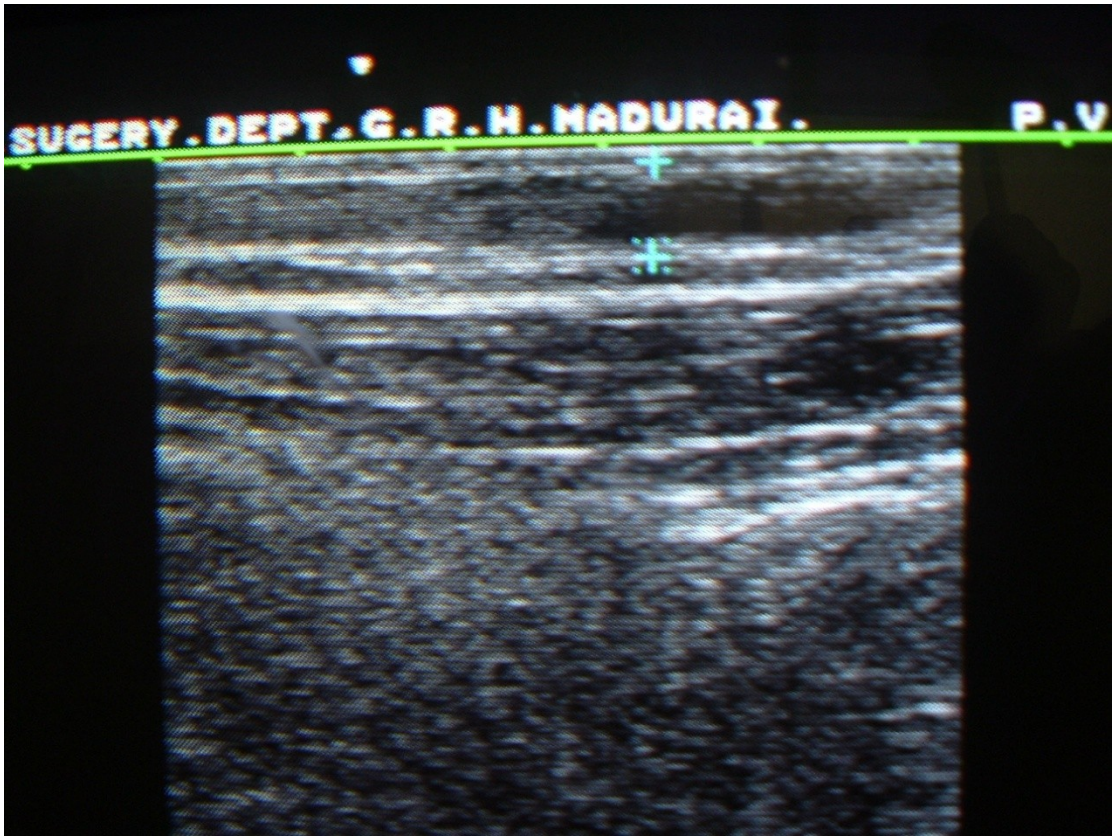
Patients in both groups were followed up for a period of 1 yr and compared for the following parameters

1. Procedure time
2. Perioperative complications

3. Complete occlusion of treated veins (by Duplex scanning at 3 months)
4. Healing of venous ulceration
5. Quality of life such as time to return to normal activity, symptom relief, and change of disease severity measured by CEAP score
6. Recurrence of varicose veins

FOAM INJECTION UNDER USG GUIDANCE

FOAM IN GSV



OBSERVATION AND RESULTS

A. Characteristics of cases studied

Table 1 : Age

Age group	Group 1- SF ligation+ Foam sclerotherapy		Group 2- SF ligation +stripping	
	No	%	No	%
31 - 40	4	16	6	24
41 – 50	10	23.3	8	23.3
51 – 60	8	16.7	8	23.3
61 – 70	3	10.0	3	10.0
Total	25	100	25	100

AGE DISTRIBUTION

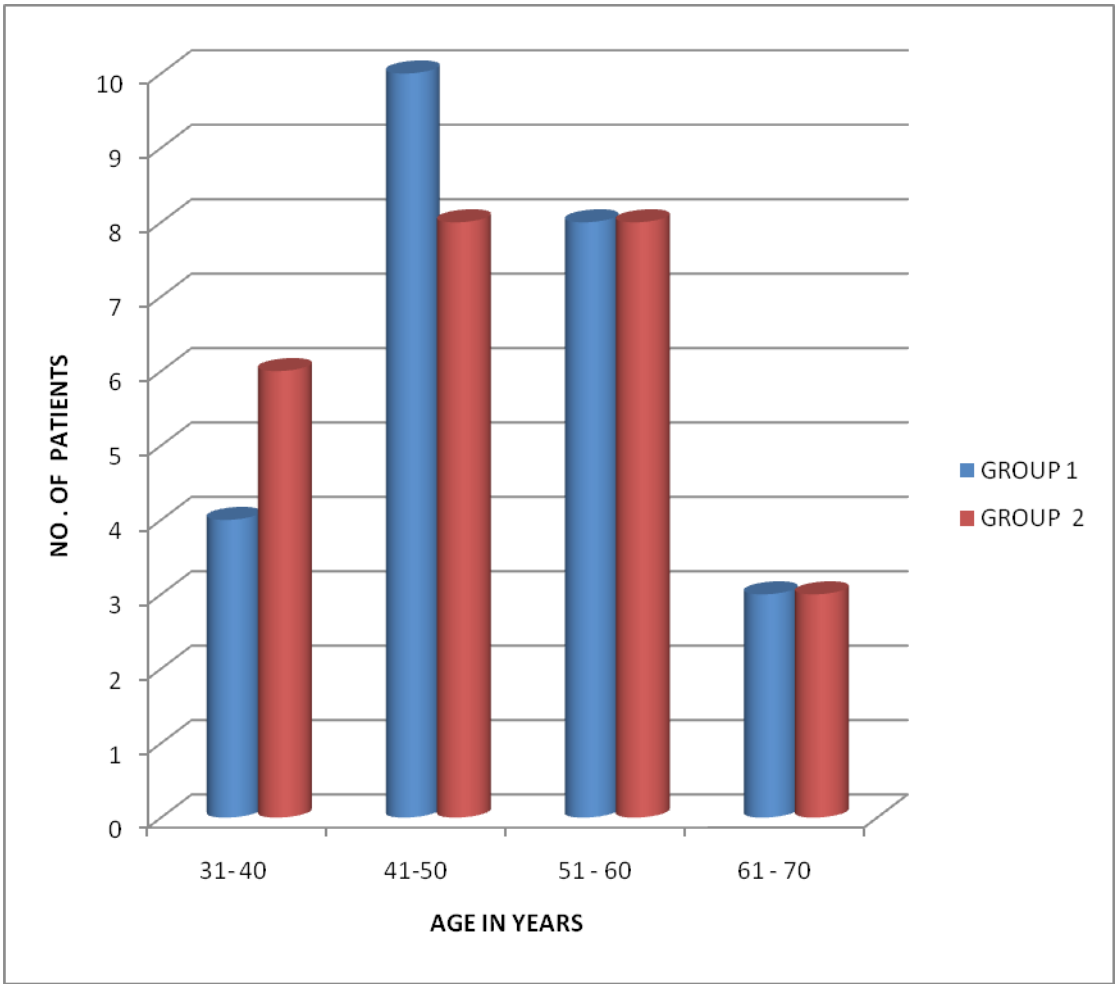


Table 2 : Sex

Sex	Group 1- SF ligation + Foam sclerotherapy		Group 2 – SF ligation + stripping	
	No	%	No	%
Males	27	90	28	93.3
Females	3	10	2	6.7

The demographic data of the patients included in this study showed no significant difference between both groups in terms of age and sex

SEX DISTRIBUTION

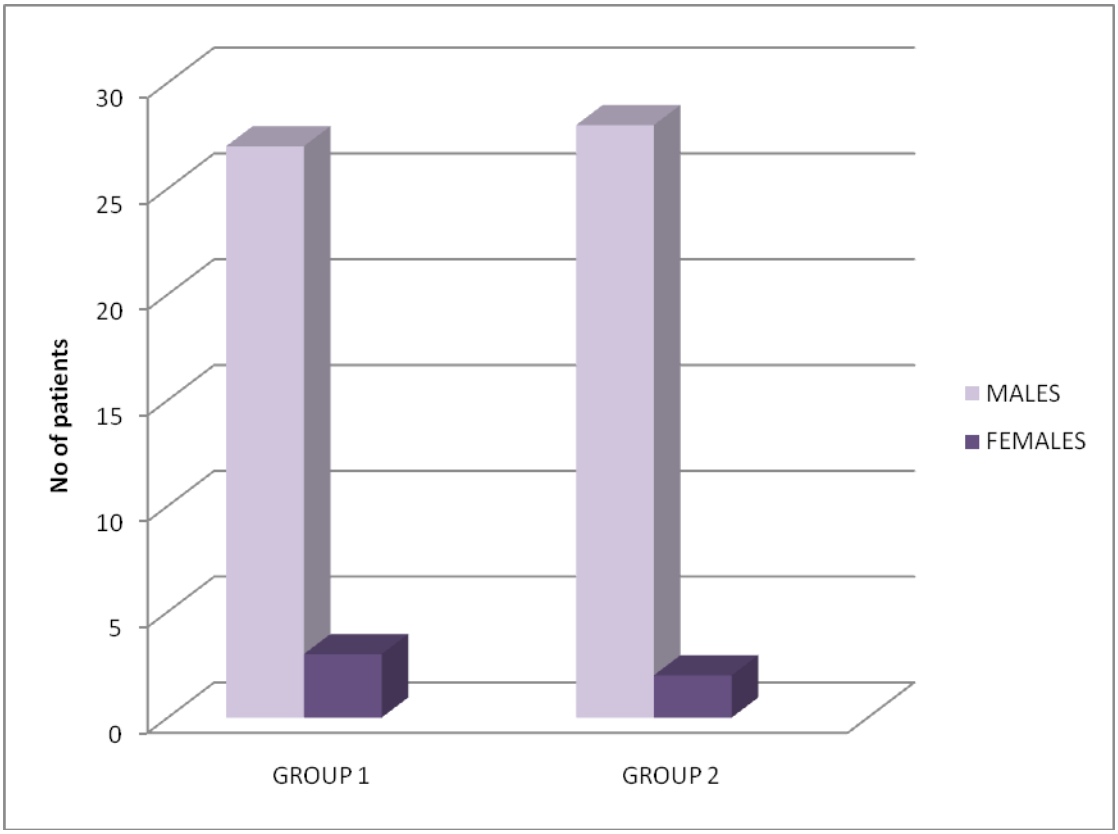


Table 3: Procedure time

Procedure time	Group 1 – SF Ligation + foam sclerotherapy		Group 2 - SF Ligation +stripping	
	No.	%	No	%
36- 45 min	19	76	3	12
46- 55 min	4	16	12	48
56- 65 min	2	8	9	36
66-75 min	-		1	4

The time taken to complete treatment was shorter in the foam sclerotherapy plus SFJ ligation group: 40 vs. 55 min

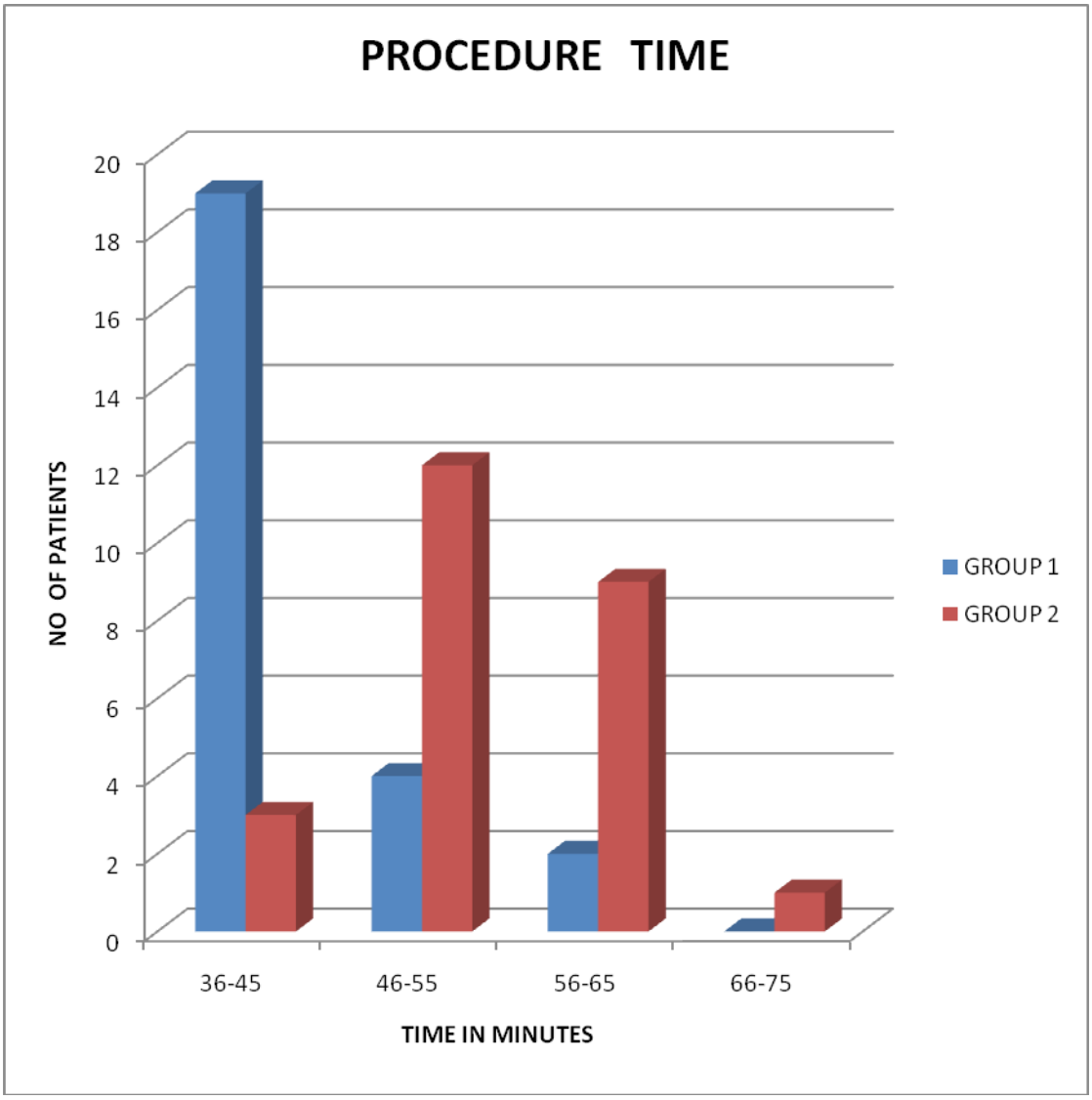


Table 4: Healing of ulcer

Ulcer healing	Group 1- SF ligation + foam sclerotherapy		Group 2- SF ligation +stripping	
	No.	%	No.	%
Healed	6	85.7	6	75
Residual ulcer	1	14.3	2	25

The rate of longer-term (>30 days) ulcer healing rate in the case series ranged from 75 to 85%. 6 (85.7%) of 7 ulcers healed in the foam sclerotherapy group compared with 6 (75%) of 8 in the SF ligation and stripping group

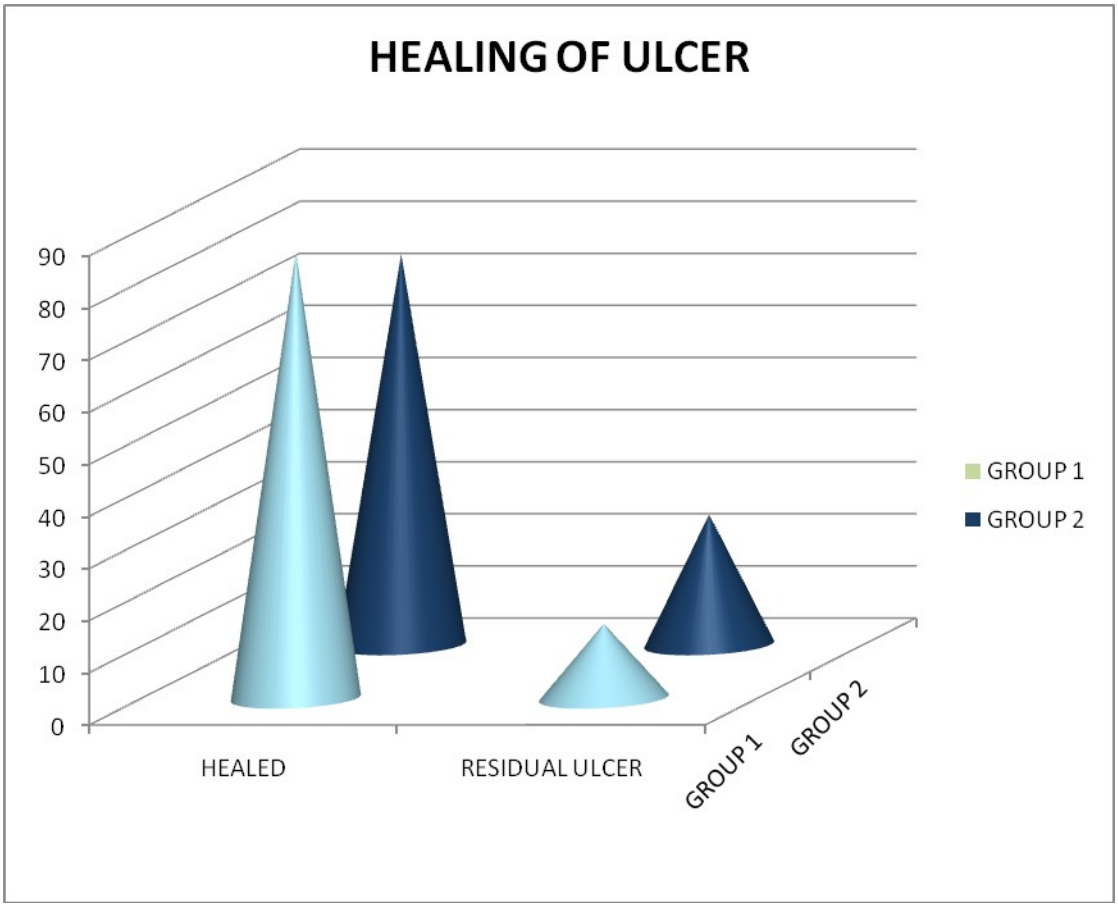


Table 5: Quality of life (CEAP score change)

C score improved by	Group 1- SF ligation + Foam sclerotherapy		Group 2 – SF ligation +stripping	
	No.	%	No.	%
1	1	4	2	8
2	7	28	9	36
3	15	60	13	52
4	2	8	1	4

At 3 months, median CEAP class dropped from four pre-operatively to one following treatment in both groups

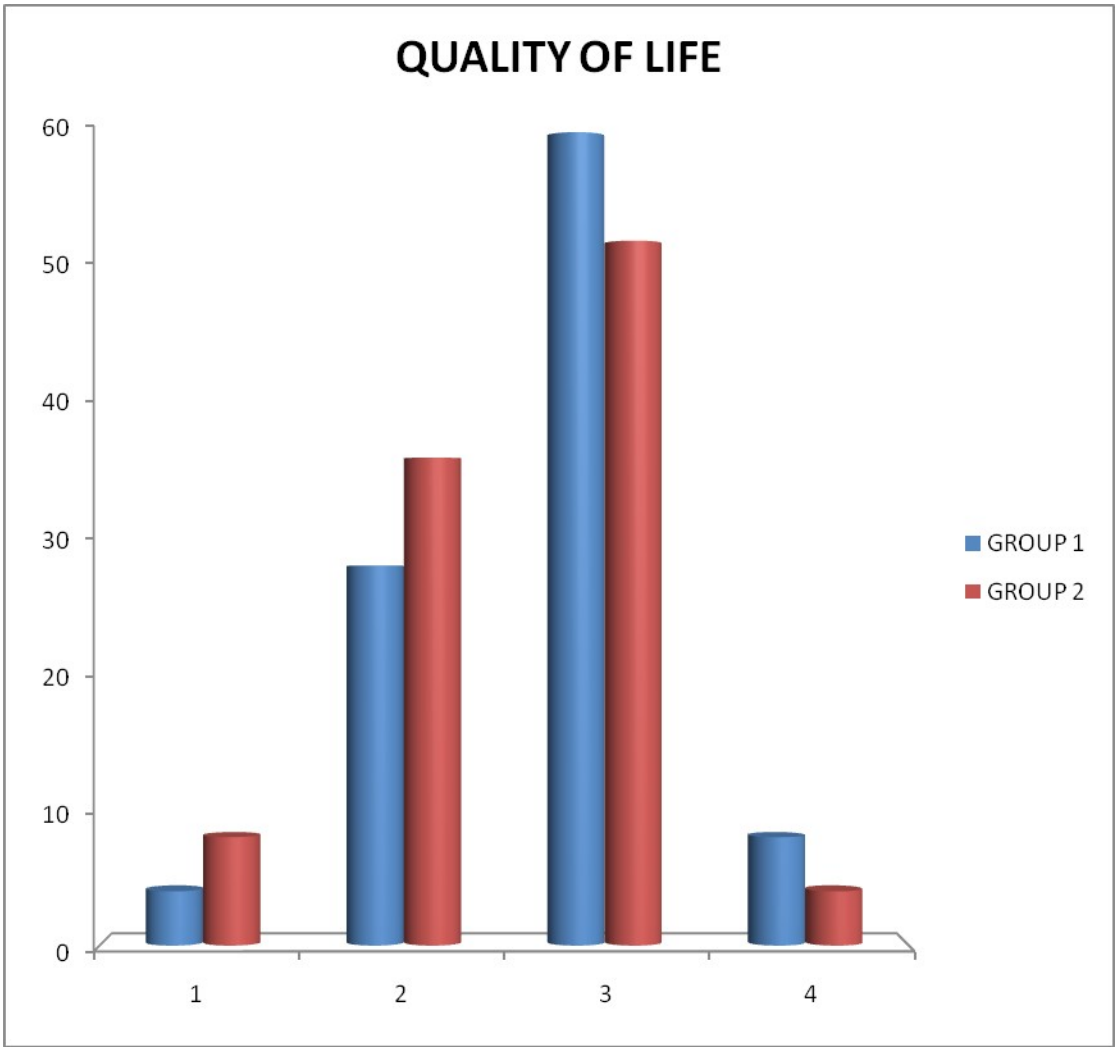


Table 6: Recurrence at 1 yr

Recurrence	Group 1 – SF ligation+ Foam sclerotherapy		Group 2 – SF ligation + stripping	
	No.	%	No.	%
Present	1	4	-	0
Absent	24	96	25	100

1 patient out of 25 treated by foam sclerotherapy reported venous recanalisation at one year after treatment. There was no recurrence in the group that underwent stripping and multiple phlebectomies.

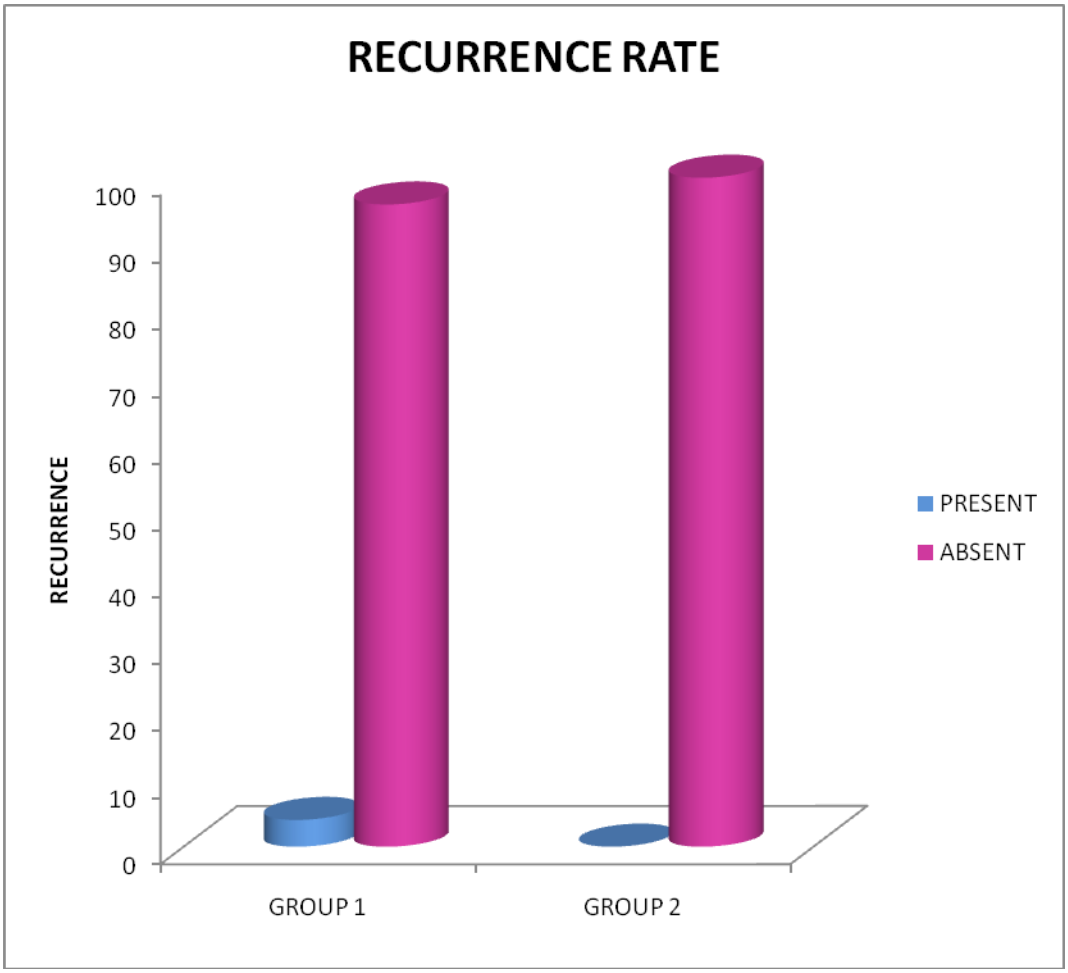


Table 6: Time to return to normal activity

Time in days	Group 1 – SF ligation+ Foam sclerotherapy		Group 2 – SF ligation + stripping	
	No.	%	No.	%
2-3	1	4	-	0
4-5	12	48	2	8
6-7	8	32	3	12
8- 9	4	16	17	68
10-11	-	0	3	12

Median time to return to normal activities was significantly reduced in the foam sclerotherapy group (4 days) compared to the surgical group (9 days)

RECOVERY TIME

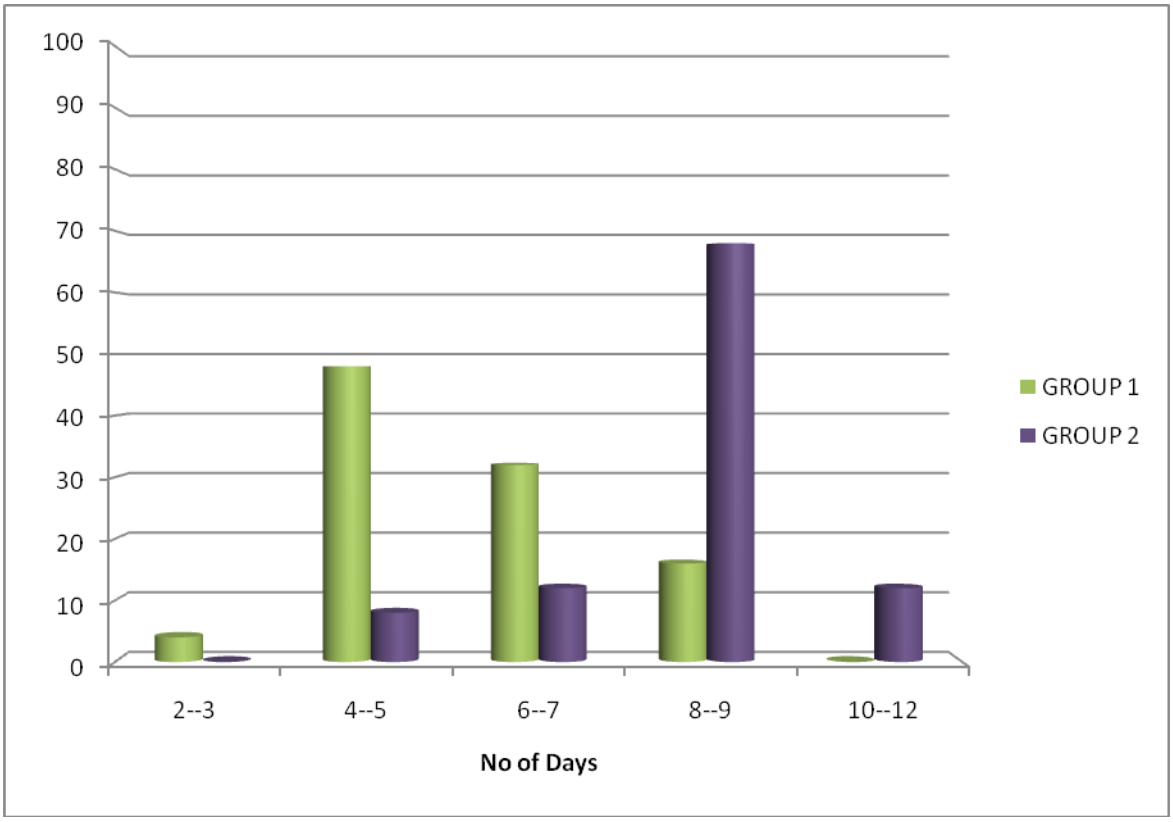
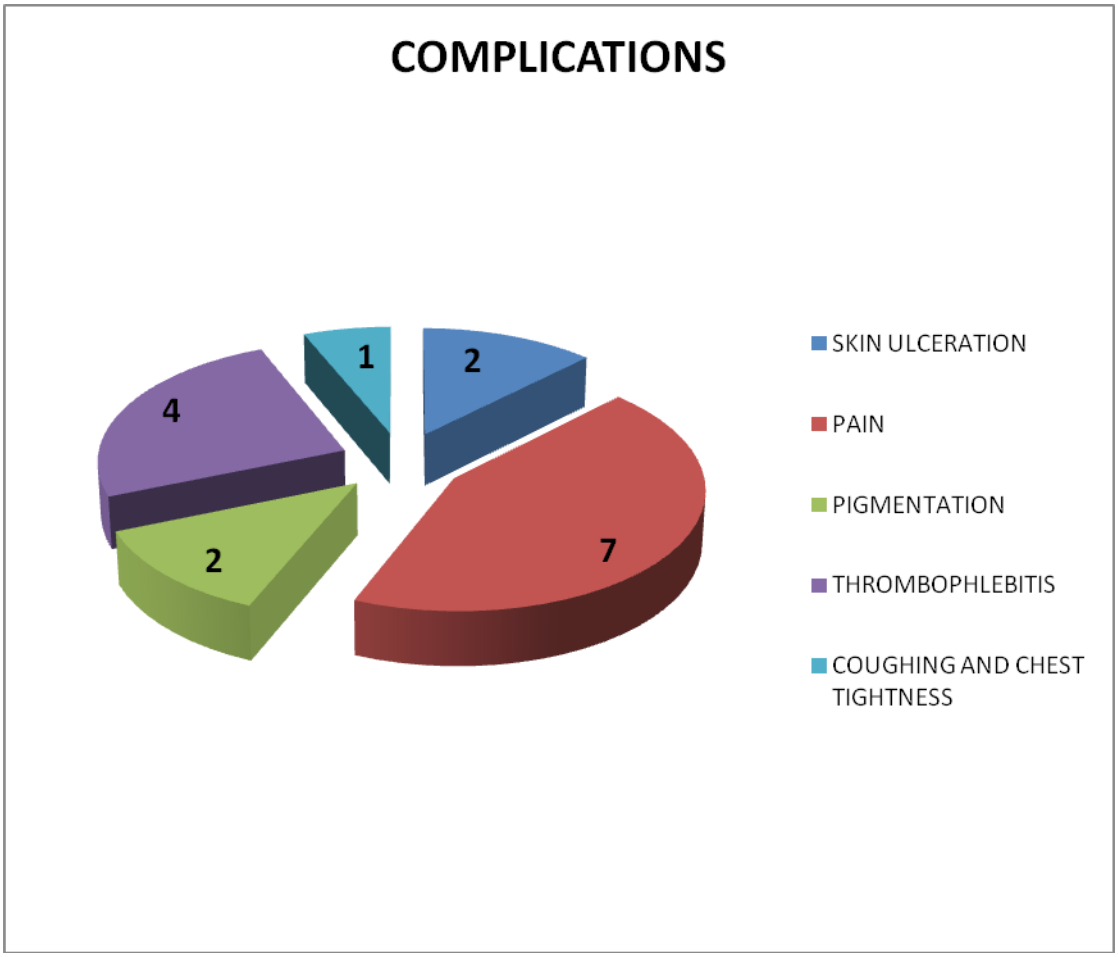


Table 7: Complications in Foam sclerotherapy

Complications in Foam sclerotherapy	No.	%

Skin ulceration	2	8
Pain at injection site	7	28
Pigmentation	2	8
Thrombophlebitis	4	16
Coughing &chest tightness	1	4

Local adverse events were relatively common. Rates of thrombophlebitis was 16%.skin staining/pigmentation at 8%, and pain at the site of injection ranged was 28%. There were no systemic complications except for a patient with transient cough and chest tightness.No serious adverse events associated with foam sclerotherapy, including arterial events like stroke and myocardial infarction, grand mal epileptic fit, pulmonary embolism, and deep vein thrombosis were encountered



VARICOSITY OF GSV

HEALED VENOUS ULCER

DISCUSSION

Significant advances have occurred in the understanding, diagnosis, and management of venous insufficiency over the last decade or so, mostly owing to the use of duplex ultrasound (DUS) technology. Stripping of the great saphenous vein (GSV) has been widely agreed upon as essential to minimizing recurrence due to redevelopment of incompetent communication with the sapheno femoral confluence and/or thigh perforator incompetence. Stripping of the vein requires additional incisions at the knee or below the knee and is associated with a high prevalence of minor surgical complications. Avulsion phlebectomy requires multiple 2- to 3-mm incisions along the course of the vein and can cause damage to adjacent nerves and lymphatic vessels.

Sclerotherapy with liquid sclerosant was introduced as an alternative to surgery but was soon abandoned as a result of high failure rates , frequent recurrence and unacceptably high rates of complications due to the large volume of sclerosant required.

Foam sclerotherapy which has gained popularity in the last decade and has been evaluated in this study as an alternative to stripping of

varicose veins. Converting the sclerosant to foam has served to reduce the dose of sclerosant and also increased its efficacy

In this study there was no significant difference in the median age and sex ratio in the case and contol groups. In the present series the time required for foam sclerotherapy and Sapheno femoral ligation was significantly less than that required for Sapheno femoral ligation, stripping and multiple phlebectomies.

Bountouroglou reported data on operation time (foam sclerotherapy plus ligation was 45 minutes versus 85 minutes for ligation plus stripping plus avulsion). The foam sclerotherapy was combined with sapheno-femoral junction ligation. this goes well with the present series.

Three case series involving 216 patients and two case reports involving three patients reported data on healing of venous ulcers. All studies used polidocanol foam. The ulcer healing rate in the case series ranged from 76.4% to 100% .In is study Sodium Tetra decyl sulphate was the sclerosant used and showed healing rates of 85%. It is unclear if the better healing rate can be atitributed to the sclerosant used.

Majority of patients in both groups in our series showed improvement in the quality of life (measured by the CEAP score) compared to baseline, however there was no significant difference between the two groups , showing that both were equally efficacious.

A RCT by Hamel-Desnos C, involving 45 patients treated by foam sclerotherapy reported venous recanalisation at one year after treatment, with a rate of 4.4%. This was lower than that in the liquid sclerotherapy group but not statistically significant (relative risk 0.5, 95% CI 0.1 to 2.5).

One non-randomised comparative study by Yamaki T involving 37 patients treated by foam sclerotherapy reported venous disease recurrence also at one year after treatment, with a rate of 8.1%, which was also lower than that in the liquid sclerotherapy group. In the present series 1 out of 25 (4%) patients showed recurrence at 1 yr.

Bountouroglou DG et al, in their study found that the median time required to return to normal activity following surgery (13 days) was significantly higher compared to those who underwent foam sclerotherapy (2days). This correlates well with our series in which most patients recovered in 8 days following surgery and in 2 days following foam sclerotherapy.

'Minor' vein thrombosis (rates ranged from 0 to 17.6%), thombophlebitis (rates ranged from 0 to 45.8%), and skin matting/pigmentation/staining (rates ranged from 0 to 66.7%), were relatively common occurrences and their incidence was similar to those in comparator groups, other than in one RCT where the risk of skin matting/pigmentation/staining was significantly higher for foam sclerotherapy compared with surgery. Pain provoked by injection or long-term pain localised at the area sclerosed was reported as ranging from 0.6 to 41.0%.

Arterial events, particularly stroke and myocardial infarction (MI), can be life threatening. One case of stroke was reported One possible explanation for arterial events is the existence of a Patent Foramen Ovale (PFO), especially with right-to-left shunt.

In our series the commonest complication encountered was pain at site of injection at 28% and thrombophlebitis (16%) followed by skin ulceration and pigmentation(8%).

CONCLUSION

To conclude, treatment of varicose veins with Foam sclerotherapy and saphenofemoral ligation or Saphenofemoral ligation and stripping provide similar results, but the use of Foam sclerotherapy instead of stripping and avulsions reduces the operative and post operative recovery time.

Sclerotherapy obviates the need for general anaesthesia and hence can be used in patients who are poor candidates for surgery.

The short period of follow up in this study is insufficient for observing longerterm efficacy. Though this study has established the safety of Foam sclerotherapy further studies with a longer follow-up period , are required to determine the comparative effectiveness of foam sclerotherapy and its optimal place in clinical practice.

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S.No	NAME	AGE	SEX	I.P NO	PROCEURE TIME	ULCER HEALING	CEAP SCORE CHANGE	RECURRENCE	RECOVERY TIME	COMPLICATIONS
1	CHANDRAN	41	M	459235	35	NO ULCER	3	NO	4	NIL
2	MUTHURAJ	54	M	413832	38	NO ULCER	2	NO	6	THROMBOPHLEBITIS + PAIN
3	MAHALINGAM	31	M	463935	40	NO ULCER	3	NO	7	NIL
4	PAPPAIAH	42	M	491900	45	HEALED	2	NO	9	PAIN
5	SAKTHIVEL	42	M	499187	50	NO ULCER	2	NO	6	PAIN
6	GOVINDAN	35	M	512495	42	NO ULCER	3	NO	7	PIGMENTATION
7	MARIAMMAL	38	F	518741	37	NO ULCER	3	NO	2	NIL
8	SUBRAMANI	41	M	526179	56	HEALED	4	NO	6	NIL
9	RAMU	55	M	529257	38	NO ULCER	3	NO	8	SKIN ULCERATION+PAIN
10	SHANMUGAM	61	M	532496	36	NO ULCER	2	NO	5	NIL
11	RAVI	39	M	535320	39	NO ULCER	3	NO	7	PAIN
12	KARTHIKEYAN	43	M	535806	40	NO ULCER	3	NO	6	THROMBOPHLEBITIS
13	PONNUTHAI	52	F	542550	48	RESIDUAL ULCER	1	NO	4	NIL
14	SIVANADI	63	M	543236	39	NO ULCER	3	NO	4	NIL

15	SAKTHIVEL	41	M	543920	40	NO ULCER	3	NO	6	THROMBOPHLEBITIS =+ PAIN
16	POTHUMPONNU	56	F	545273	42	HEALED	3	NO	4	NIL
17	PERUMAL	46	M	545863	36	HEALED	4	NO	4	NIL
18	PITCHAI	63	M	548375	38	NO ULCER	3	NO	9	SKIN ULCERATION+PAIN
19	PANDIAN	45	M	548649	40	NO ULCER	2	YES	4	NIL
20	RAMAR	55	M	549277	50	HEALED	3	NO	5	PIGMENTATION
21	VELLAISAMY	57	M	549329	43	NO ULCER	2	NO	4	NIL
22	MURUGESAN	53	M	550620	46	HEALED	2	NO	8	THROMBOPHLEBITIS
23	MARIYAPPAN	45	M	588132	40	NO ULCER	3	NO	4	NIL
24	MUTHUMAYAN	56	M	578456	37	NO ULCER	3	NO	4	NIL
25	RAHEEM BASHA	42	M	578562	38	NO ULCER	3	NO	4	NIL

S.NO	NAME	AGE	SEX	I.P.NO	PROCEDURE TIME	ULCER HEALING	CEAP SCORE CHANGE	RECURRENCE	RECOVERY TIME

1	ABDUL KADHAR	45	M	459235	48	NO ULCER	2	NIL	8
2	DHARMARAJ	53	M	463935	55	HEALED	3	NIL	9
3	ALEXPANDIAN	34	M	413832	57	NO ULCER	2	NIL	6
4	DEVARAJ	55	M	482888	60	NO ULCER	3	NIL	9
5	VARADHARAJAN	63	M	486200	55	NO ULCER	3	NIL	9
6	SOLAI	57	M	488215	60	HEALED	2	NIL	11
7	KANNAN	45	M	493534	53	HEALED	2	NIL	8
8	VELU	56	M	491877	54	RESIDUAL ULCER	1	NIL	10
9	RAKKU	36	F	513209	40	NO ULCER	3	NIL	7
10	ANTONY	43	M	513247	52	NO ULCER	3	NIL	8
11	MUTHUSAMY	61	M	526320	63	NO ULCER	2	NIL	9
12	RATHINAM	35	M	516212	55	HEALED	3	NIL	8
13	MUNIYANDI	55	M	539391	64	NO ULCER	3	NIL	7
14	PUSHPAM	47	F	541543	49	NO ULCER	2	NIL	9
15	MURUGAN	52	M	541552	58	HEALED	3	NIL	8
16	MARIYAPPAN	55	M	541749	70	NO ULCER	3	NIL	9
17	RAJKUMAR	35	M	545260	48	HEALED	4	NIL	4
18	PANDIARAJAN	45	M	548738	57	NO ULCER	2	NIL	8
19	SOLAIMALAI	31	M	550545	55	NO ULCER	3	NIL	9
20	MUSTAFA	62	M	552026	48	NO ULCER	3	NIL	5
21	MUTHUPANDI	52	M	552823	42	RESIDUAL ULCER	1	NIL	10
22	MANI	48	M	553272	60	NO ULCER	2	NIL	8

23	PALANIKUMAR	37	M	553603	45	NO ULCER	2	NIL	8
24	SELVAKUMAR	45	M	553889	55	NO ULCER	3	NIL	9
25	SEKARAN	41	M	554121	58	NO ULCER	3	NIL	9